Acute aluminium phosphide poisoning, what is new? Singh Yatendra, Joshi Subhash C., Satyawali Vivekanand, Gupta Abhisek

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Aluminium phosphide (AIP) is a cheap solid fumigant and a highly toxic pesticide that is commonly used for grain preservation. AIP has currently generated interest with increasing number of cases in the past four decades because of its increased use for agricultural and nonagricultural purposes, and also its easy availability in the markets has led to its increased misuse to commit suicide. Ingestion is usually suicidal in intent, uncommonly accidental and rarely homicidal. The poison affects all systems, shock, cardiac arrhythmias with varied ECG changes and gastrointestinal features being the most prominent. Diagnosis is made on the basis of clinical suspicion, a positive silver nitrate paper test to phosphine, and gastric aspirate and viscera biochemistry. Treatment includes early gastric lavage with potassium permanganate or a combination of coconut oil and sodium bicarbonate, administration of charcoal and palliative care. Specific therapy includes intravenous magnesium sulphate and oral coconut oil. Unfortunately, the lack of a specific antidote results in very high mortality and the key to treatment lies in rapid decontamination and institution of resuscitative measures. This article aims to identify the salient features and mechanism of AIP poisoning along with its management strategies and prognostic variables.

Keywords:

aluminium phosphide, management, phosphine, poisoning, shock

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Introduction

Aluminium phosphide (AIP) is a solid fumigant that has been used extensively since the 1940s. It is easily available and can be purchased in some countries such as India under the trade names, for example Celphos, Quickphos, Synfume and Phosfume [1]. AIP is a solid pesticide that rapidly became one of the most commonly used grain fumigants because of its properties, which are considered to be near ideal; it is toxic to all stages of insects, highly potent, does not affect seed viability, is free from toxic residues and leaves little residue on food grains [2]. This highly toxic chemical is cheap and usually formulated in tablets or pellets, granules and as dust. Upon contact with moisture in the environment, it undergoes a chemical reaction, yielding phosphine gas, which is the active pesticidal component [3].

No reports are available in the English literature from any part of the world where AIP has been ingested as a poison to commit suicide, except in Morocco (1995), where death rates by suicides by self administration of AIP pills are high [4], and in Denmark (1996), where a case of AIP ingestion has been reported. During the last two decades, poisoning because of AIP ingestion has been reported [5] widely from different Northern States of India, where the AIP poisoning was unknown before 1980. The first case was reported in 1981, but since then, the number of cases has been increasing progressively throughout North India so much so that it is now the single most common suicidal method and the problem has acquired an epidemic proportion [6–8]. It has currently generated interest with increasing number of cases in the past four decades because of its increased use for agricultural and nonagricultural purposes; also, its ease of availability has led to its increased misuse to commit suicide [9,10].

Aluminium phosphide

AlP is a solid fumigant and ideal pesticide since 1940 as it is cheap, most efficacious and easy to use and freely available over the counter in India (as Alphos, Celphos, Quickphos, Phostek, Phosfume and Synfume) and in Morocco (as Phostoxin) in the form of chalky white or brown 3 g tablets containing 56% of AlP and 44% of ammonium carbonate [4,11,12]. The tablets are taken out of a sealed container and placed on stored grains and the storage container is closed for a few days to eliminate moles and vermines in granaries [4]. AlP has a relatively high vapour pressure, which allows it to penetrate porous material effectively. On coming into contact with water or moisture or OH radical of air or hydrochloric acid in the stomach, a 3 g tablet of AlP releases 1 g of phosphine or phosphorus hydrogen [12], that is–

 $AlP + 31-1,0 = Al(OH) + PH_{3}.$

 $AIP + 3HC1 = AIC1 + PH_3$.

Phosphine (PH₃)

PH is a colourless gas with a fishy or garlic odour with an odour threshold of 0.14 ppm and a vapour density of 1.17 [13,14]. It has a half-life of 5–24 h, which is dependent on the surface and the permeability of the solid matrix. The nontoxic residues, that is phosphites and hypophosphites of aluminium left in the grains, are less than 0.1 mg. PH (1 kg), for raw cereals, is the WHO-recommended permissible level considered safe for human consumption [12].

Mechanism of action

After oral intake, the phosphine gas released is absorbed by the gastrointestinal tract by simple diffusion. Phosphine, like cyanide, inhibits mitochondrial cytochrome oxidase and cellular oxygen utilization. Phosphine can inhibit cytochrome C oxidase *in vitro*, but it has much less activity *in vivo* [15–18]. This inhibits oxidative respiration by 70% and ultimately results in a marked decrease in mitochondrial membrane potential [16].

In the presence of AIP, cellular superoxide and peroxide radicals are generated, with subsequent cellular damage by lipid peroxidation [19–22]. The higher levels of superoxide dismutase, malonyldialdehyde and catalase in postmortem studies and decrease in the serum levels of these biochemical parameters to a normal level in survivors indicate their role in poisoning [23]. Some experimental results suggest the beneficial role of glutathione, melatonin, vitamin C and carotenes [24].

The direct toxic effects of phosphine on cardiac myocytes, fluid loss and adrenal gland can induce profound circulatory collapse [18]. Phosphides and phosphine can exert a direct corrosive effect on body tissues [25].

Toxicity

The fatal dose in an adult is 150–500 mg [10,26]. The permissible exposure limit is 0.3 ppm over an 8 h shift (for factory staff) [27].

Epidemiology

In the largest series comprising of 418 cases reported from Rohtak, India, the hospital incidence of AlP poisoning was 0.06/1 000 hospital admissions, which increased progressively to 10/1000 in 1989–1990, with a male female ratio of 2 : 1, and is now increasing continuously so as to surpass any other poisoning in Haryana [6,7]. In Washington State, AlP poisoning comprised 38.42% (15 out of 39 cases) of fumigantrelated illnesses [14]. Stray incidences of AlP ingestion have been reported from Morocco and Denmark [4,5].

AlP poisoning is more common in the rural belt of North India, where the agricultural community, irrespective of sex, is more at risk, which correlates positively with illiteracy, frustration, depression, failure in examination, disputes in the family, inability to find suitable avenues of income and easy availability of AlP in the household [8,28]. The occupational threshold value of pH is 0.3 ppm [14] and a concentration of more than 7 ppm in air can cause serious illness; a concentration of 290–300 ppm is life threatening; 400– 600 ppm is lethal in half an hour; and a concentration of 1000 ppm is rapidly fatal. The fatal dose after ingestion is 150 mg for a person weighing 70 kg and the fatal period is 1–96 h, the average being 28 h [6–8].

Clinical manifestations

Vomiting, abdominal pain, loose motions and restlessness are frequent presenting complaints. Cardiovascular involvement results in thready pulse, tachycardia, tachypnoea, acidosis, marked hypotension, palpitation and ultimately unresponsive shock. Patients remain mentally lucid until cerebral anoxia because of shock develops. AIP poisoning may have complications such as haemorrhage, acute renal failure, disseminated intravascular coagulation and arrhythmias. Several ECG changes ranging from ST segment elevation/ depression, PR and QRS interval prolongation, complete heart block to ectopic pace making and also fibrillation have been reported [29].

Patients can present with anteroinferior wall ischaemia, right bundle branch block and T-wave flattening/ inversion simulating myocardial ischaemia. These changes are because of toxic injury to myocardium [30]. Pulmonary oedema, dyspnoea, cyanosis and altered sensorium may be present in AlP poisoning. Other rare effects include hepatitis, disseminated intravascular coagulation and acute tubular necrosis [26,31].

Laboratory investigations

Laboratory evaluation is mainly performed to assess the prognosis. Leucopenia indicates severe toxicity. Increased SGOT or SGPT and metabolic acidosis indicate moderate to severe ingestional poisoning. Electrolyte analysis shows decreased magnesium, whereas potassium may be increased or decreased [32].

Measurement of plasma renin is significant as its level in blood has a direct relationship with mortality and is increased in direct proportion to the dose of pesticide. The serum level of cortisol is usually found to be decreased in severe poisoning [33]. Chest radiograph may indicate hilar or perihilar congestion if acute respiratory distress syndrome (ARDS) develops.

ECG shows various manifestations of cardiac injury (ST depression or elevation, bundle branch block, ventricular tachycardia, ventricular fibrillation) [29,34,35]. Wall motion abnormalities, generalized hypokinesia of the left ventricle, decreased ejection fraction and pericardial effusion can be observed in echocardiography [36].

Diagnosis

Diagnosis is made on the basis of clinical suspicion, history of inhalation exposure or history of ingestion of AIP, corroborated by seeing the tablets of AIP or its empty container left in the house, clinical manifestations with garlic or decaying fish odour, unexplained shock, a positive $AgNO_3$ test, chemical analysis of gastric juice and viscera and histopathological changes [37–39].

Mortality

The specified fatal dose of AlP is 0.15–0.5 g. However, most of the patients present with ingestion of three or more tablets, which invariably results in death [2]. The average time interval between ingestion of AIP and death is 3 h (1-48 h), 95% of the patients die within 24 h and the most common cause of death in this group is cardiac dysrhythmia. The severity of poisoning depends on the type of compound consumed. Fresh and active compounds (tablets) commonly affect the heart, lungs, gastrointestinal tract and kidneys, causing severe metabolic acidosis and high mortality. Broken or granular forms of tablets cause mild hypotension and ECG changes, mild metabolic acidosis and low mortality as the activity of the compound is less. The powder form of tablets is inactive, and causes no systemic effects and no mortality [40]. Death after 24 h occurs usually because of shock, acidosis, ARDS and cardiac dysrhythmia. Mortality depends on the dose of poison, severity of poisoning, duration of shock, failure of response of shock to resuscitative measures and severity of hypomagnesaemia [41]. Nonsurvivors have more severe hypotension and metabolic acidosis than the survivors who have more severe vomiting [31]. The mortality rate is highly variable, ranging from 37 to 100%, and can reach more than 60% even in experienced and well-equipped hospitals [2].

Management

General

The management of AlP poisoning should be initiated as soon as possible and should not be delayed on confirmation of poisoning [42]. Gastric lavage is performed with potassium permanganate (1 : 10 000) to reduce the absorption of phosphine. Potassium permanganate (1 : 10 000) oxidizes PH_3 to form nontoxic phosphate. This is followed by activated charcoal (~100 g) administered through a nasogastric tube. Antacids and proton pump blockers are added for symptomatic relief [43,44]. It is reported that lavage with coconut oil also plays a role in the management of acute AlP poisoning even 6 h after ingestion. Correction of plasma glucose levels may be useful in patient management [45,46].

The most important aspect is resuscitation of shock. An intravenous line should be established as soon as the patient arrives and 2-3 l of normal saline should be administered in the initial hours (8-12) guided by central venous pressure and pulmonary capillary wedge pressure. The aim is to maintain the central venous pressure at around 12-14 cm of water [47]. Some reports have recommended rapid infusion of saline (3-6 l) in the initial 3 h. Low-dose dopamine (4-6 µg/kg/min) is administered to maintain systolic blood pressure greater than 90 mmHg. Hydrocortisone 200–400 mg every 4–6 h is administered intravenously to combat shock, reduce the dose of dopamine, check capillary leakage in lungs (ARDS) and to potentiate the responsiveness of the body to endogenous and exogenous catecholamines [24,48,49]. Oxygen is administered for hypoxia. Phosphine excretion can be increased by maintaining adequate hydration and renal perfusion with intravenous fluids and lowdose dopamine (4–6 μ g/kg/min). Diuretics such as furosemide can be administered if systolic blood pressure is greater than 90 mmHg to enhance the excretion of phosphine [50]. All types of ventricular arrhythmias can be observed in these patients and the management is the same as for arrhythmias in other situations [50]. Bicarbonate level less than 15 mEq/l requires sodabicarb at a dose of 50–100 mEq intravenously every 8 h until the bicarbonate level increases to 18-20 mEq/1 [36].

Specific therapy

There is no specific therapy for this poisoning. The management of intoxication from AlP is mainly supportive. The administration of H_2 receptor antagonists or proton pump inhibitors has been recommended to reduce the gastric acidity and further release of phosphine gas. Hypomagnesaemia is a common outcome of phosphine gas exposure and intracellular magnesium plays an important role as a cofactor in the synthesis of several antioxidants; thus, administration of intravenous magnesium has been investigated in cases of AlP poisoning [51].

In a trial of 50 patients, individuals receiving repeated doses of intravenous magnesium showed significant improvement in indicators of oxidative stress and a lower incidence of mortality (20%) in comparison with control participants (44% mortality) [52]. Oral administration of the anti-ischaemic drug trimetazidine, which works through a metabolic mechanism of decreasing the production of oxygen-derived free radicals and stimulating the oxidative metabolism of glucose, has been suggested. It was temporally associated with clinical improvement in a case report of occupational inhalation exposure to phosphine gas from AIP [53–55]. The use of magnesium sulphate (both high and low dose) did not improve survival in controlled clinical trials. Hence, its use is not recommended [56]. Administration of sorbitol solution (at a dose of 1–2 ml/kg) as a cathartic and vegetable oils and liquid paraffin as an inhibitor of phosphine release from the overdosed AIP has been suggested [57]. Digoxin has been suggested for the treatment of cardiogenic shock induced by acute AIP intoxication [58].

Conclusion

Exposure to phosphine gas released from AIP fumigants increases the risk of major morbidity and mortality. There is an increasing need for improved knowledge of these risks, with an emphasis on recognition, management and prevention.

The mortality because of AlP poisoning is very high and variable, and depends on a number of factors, the lack of an antidote and the poor prognostic signs being the most prominent. The use of MgSO, to reduce cardiac arrhythmias and mortality, is well documented and recommended. The fast progression to lifethreatening symptoms, ineffective therapeutic ways to counter its intoxication and limited data on the efficacy of therapeutic interventions pose challenges to the clinicians and emergency staff.

The most important factor that may help to improve survival is providing preliminary medical aid within 0.5–1 h of AlP intake at grassroot levels. Other preventive measures could be the caging of tablets in plastic packs with holes and spikes and more stringent restrictions on its supply in open market. The applicators of AlP must be licensed or should work under the supervision of a licensed individual. Improved education and enforcement of safety regulations would help to reduce the frequency of the illness. Thus, the problem needs to be tackled by using a multifaceted approach in the form of preventive measures, updating the management modalities at all levels and overall, study of and research for an antidote that can prevent further loss of human lives as a result of poisoning.

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